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Multiple sclerosis brain lesion measurements in clinical practice

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Accurate detection of brain lesions in multiple sclerosis (MS) patients is important for diagnosis and measuring therapeutic response. In clinical practice, lesion load is often visually inspected or quantified by manual or semi-automated segmentation of Magnetic Resonance Images (MRI), which is time-consuming, costly, and associated with large inter- and intra-observer variability. We propose an automated lesion segmentation method, with high reliability and accuracy. In this approach, 2D or 3D T1-weighted and FLAIR MR images are used to classify the brain into GM, WM and CSF. In addition, by using a healthy brain atlas, MS lesions are detected as an outlier to the normal brain. The method is evaluated on the brainWeb's MS simulated dataset. For evaluation, three types of MS patients are considered, namely, mild, moderate and severe. In case of mild patient, the average overlapping of the lesion segmentation with the ground truth is 55.48%, 85.07% in moderate and 83.67% in severe. The average lesion volume difference between the segmentation and the ground truth in the mild case is 3.41%, 0.68% in the moderate and 2.19% in the severe. As a result, the number and volume of brain lesions is measured and can be followed-up in clinical practice. In addition, the number and volume of the brain lesions is quantified for different brain regions, such as the frontal lobe, midbrain, parietal lobe, etc. Clinicians can then relate lesion volume changes as well as the number of new lesions in different brain regions with changes in the clinical situation.

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How to increase the detection rate of chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids (CLIPPERS)

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Objectives: Chronic Lymphocytic Inflammation with Pontine Perivascular Enhancement Responsive to Steroids (CLIPPERS) is an inflammatory CNS disorder characterized by
1) sub-acute onset of cerebellar and brainstem symptoms,
2) mainly peripontine contrast-enhancing perivascular lesions with a "salt-and-pepper" appearance on MRI, and
3) angiocentric, predominantly T-lymphocytic infiltration as revealed by brain biopsy.
Neuroinfectious diseases, CNS lymphoma and, of note, neurosarcoidosis must be excluded. As CLIPPERS has been described as recently as 2010, many patients may have been misdiagnosed in the past.
Methods: We searched the medical records from the Department of Neurology at Rigshospitalet, Copenhagen University Hospital, for patients discharged between 1999-2013 with a diagnosis of "sarcoidosis with other localization" (D86), "other acute disseminating demyelination" (G36), "other demyelinating disease in the CNS" (G37) or "encephalitis, myelitis or encephalomyelitis" (G04.9).
Results: Of 206 identified patients, 24 had been examined by brain biopsy and were included for further evaluation. Following clinical, neuroradiological and neuropathological review, 3 patients (12.5%) were reclassified as having CLIPPERS. To the authors' knowledge these are the first reported Scandinavian cases of CLIPPERS; median long-term follow-up was 75 months.
Conclusions: The present results suggest that clinical review of patients previously diagnosed as neurosarcoidosis or unspecified inflammatory demyelinating CNS disease might increase the detection rate of CLIPPERS.

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